W. APPROACH TO LOWER ABDOMINOPELVIC | PAIN

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ACUTE AND CHRONIC PELVIC PAIN

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INTRODUCTION

One of the greatest challenges for the primary care physician, gynecologist, and surgeon is the diagnosis of acute onset pelvic and lower abdominal pain. When the cause is not obvious patients are evaluated by ultrasound, CT scans, and MRIs, observed to re-evaluate for developing signs and symptoms, treated empirically with antibiotics and narcotics, and, out of frustration, often questioned about drug-seeking or emotional instability. As the patient becomes increasingly anxious, the pain symptoms magnify and provider/patient communications deteriorate. While pelvic visceral pain can be complex, the clinical assessment can, as described in this chapter, help to separate patients with acute pelvic visceral lesions from those with acute pain resulting from pelvic visceral neuropathy. The latter is the neurological basis for the diagnosis of chronic pelvic pain, interstitial cystitis, vulvar vestibulitis syndrome, pelvic dyssynergia, and myofascial abdominal pain.

NEUROPATHOLOGY OF PELVIC PAIN

The **innervation of pelvic viscera** includes the autonomic nerves with the sympathetic nerves primarily originating in T_{10}-L_1 dermatomes, passing along the aorta through the superior hypogastric plexus at the bifurcation, being carried to the inferior hypogastric plexus alongside the uterine cervix by the uterosacral ligaments and innervating the uterus, tubes, bladder, ureters, cervix, upper 2/3 vagina, pelvic peritoneum. and muscles. The parasympathetic nerves originate in the S_2-$_4$ dermatomes, innervating the perineum S_2 through the pudendal nerve and deep pelvic viscera $S_{3,4}$ through the pelvic nerve and the inferior hypogastric plexus.

Noxious pain messages are carried by C fibers that physiologically are only able to result in an ache sensation regardless of the type of noxious stimulus (polymodal). C fibers, however, can be sensitized by peripheral activators and sensitizers such as gonorrhea endotoxin and tissue neurotransmitters such as bradykinins, leukokinins, histamine, serotonin, potassium, and substance P. With sensitization, the poorly localized ache sensation can become sharp, localized, and severe. The severe alteration of peripheral threshold can be observed with central sensitization either in the absence of a peripheral lesion originating from the dorsal horn or in the presence of chronic nerve stimulus, as might be seen with endometriosis, pelvic nerve trauma, or infiltrating cancer.

Peripheral visceral sensitization will alter dorsal horn threshold modulation resulting in increased visceral sensitivity as well as projected referred pain in the somatic referral area for that visceral tissue. For instance, the appendix refers to McBurney's point, midway between the umbilicus and the iliac crest. The ureter refers to the groin and lateral lower abdomen and the uterus refers to mid-lower abdomen and low back over the low lumbar and sacral spine. Projected referred pain, however, cannot be reproduced by pressure over the somatic referral area or blocked by local anesthetics.

Central visceral sensitization on the other hand produces the same visceral sensitivity to exam with the exception of rebound peritoneal pain and it differs by causing normal physiologic functions such as menses, ovulation, full bladder, full bowel, and coitus to become painful. The pathognomonic sign, however, is that central visceral sensitization (neuropathy) results in sensitization of the somatic referral area. Consequently, the somatic tissue becomes not only a major source of pain but also results in further central sensitization, potentiating the pain syndrome. In theory, this is caused by retrograde release of substance P into the somatic referral area.

ACUTE VISCERAL DISEASE

The diagnosis of pelvic disease requires close attention to common signs and symptoms at the same time the understanding that pain often precedes the development of fever, elevated WBC's in patients with appendicitis and gonorrhea salpingitis, and dropping Hct in patients with ruptured ectopic pregnancy. The most valuable sign of acute visceral disease is rebound tenderness, as it is rarely found in chronic pelvic pain. While ruptured appendix can present with nausea and vomiting, fever, elevated WBC's, and rebound, the diagnosis is helped by ultrasound/CT scan of the appendix to look for inflammation and by the progression of symptoms with close observation. Gonorrheal salpingitis can be diagnosed by fever, elevated WBC's, guarding and rebound, and gram stain. It often requires culture and at times diagnostic laparoscopy to make the diagnosis. Ectopic pregnancy is best diagnosed by a lack of doubling of the hCG over 48 hours and the absence of fetal tissue in utero on ultrasound or D&C. While adnexal masses seen on ultrasound and found on pelvic exam can be helpful, fluid in the cul de sac on ultrasound (or blood on culdocentesis) can also clarify the stage of rupture of the tubal pregnancy. Torsion of the ovary may present with intermittent pain before the acute infarction of the twisted adnexa. However, it should always be associated with the finding of a tender adnexal mass on pelvic exam or ultrasound.

Brewer ('72) evaluated 1,000 patients presenting to the emergency room with acute abdominal pain and followed up the outcome of the evaluation and treatment as to diagnosis. The single most common cause found in 41.3% of the patients was pain of unknown origin. The next most common was gastroenteritis in 6.9%, PID in 6.7%, and UTI in 5.2%.

Thomson ('67), using a technique called the abdominal wall test first reported by Carnet in 1926 (Carnet's sign), found that 23 out of 120 patients seen in the ER with acute abdominal pain in follow-up had no obvious cause. All had a positive abdominal wall test defined as the same or increased pain on abdominal palpation conducted with the rectus muscles tensed by elevating the shoulders or legs. Of the other 97 patients found to have acute visceral disease such as appendicitis 65%, mesenteric adenitis 6%, other 23%, only one had a positive abdominal wall test. The others had no pain with tension of the rectus blocking off the visceral organs. The abdominal wall then becomes not only a major source of pain but also a valuable sign of acute pain with no obvious cause or visceral neuropathy.

PELVIC VISCERAL NEUROPATHY

In order to differentiate acute visceral disease from visceral neuropathy, attention during the examination of the patient must include the separation of the viscera from the abdominal wall by tensing the rectus (Carnet's sign). The technique is as follows:

- **The abdominal exam**

    1. Have the patient point with one finger to where it hurts.

    2. Press with one finger over the area of pain, marking each point where pain is reproduced.

    3. Tense the rectus muscles by having the patient elevate her shoulders or ankles and re-examining the abdomen with one finger. If pain is reproduced with the muscles tensed, then there is abdominal wall neuropathic pain. If there is blocking of the tenderness by tensing the muscles, then the patient has visceral pain and possibly acute visceral disease.

    4. In patients with abdominal wall pain, each mark should be needled (#22) in order to reproduce the pain where the needle reaches Camper's fascia and then blocked by injecting 1-2 cc of ¼% Bupivacaine (up to total of 50 cc). Injection of the triggerpoints should result in complete relief from pain for a duration longer than the action of the local anesthetic (4-6 hours). Typically, patients notice days to months of relief.

5. Patients should return for re-evaluation in 2-4 weeks and be re-injected 3-4 times if triggerpoints return to look for prolonged relief.

6. Abdominal wall triggerpoints found in Camper's fascia (not rectus muscle) can be found in 96% of patients with chronic pelvic pain and interstitial cystitis.

- **The pelvic exam**: finding that the abdominal wall accounts for a major part of chronic pelvic pain still doesn't explain the increase in pain with normal pelvic visceral function. It is critical that all pelvic viscera and structures be examined independently. This cannot be done by the standard bimanual pelvic exam. The technique is as follows:

    1. Examine the Bartholin's glands, Skene's glands, and posterior fourchette with a Q-tip to rule out vulvar vestibulitis syndrome (VVS). VVS is a neuroinflammatory pain syndrome presenting as dyspareunia (burning after, and sharp knife like pain during, coitus) and clinically presents as erythema around the Bartholin's and Skene's gland orifices and sharp, stabbing tenderness to Q-tip pressure. The insertion of a speculum often induces sharp VVS pain.

    2. Examination of the levator and obturator muscles is done by single finger lateral pressure just inside the vagina (levators) and deep lateral pressure while the patient abducts the knee against your external hand to tense the obturator internus muscle. Muscle tenderness without injury or inability to relax the muscles is a part of the sacral neuropathy associated with interstitial cystitis.

    3. Examination of the bladder must precede the bimanual examination of the uterus and ovaries because the vaginal fingers push through the bladder. Bimanual compression of the bladder in interstitial cystitis and chronic pelvic pain not only is extremely painful, but it also reproduces the patient's same pain. 86% of women with interstitial cystitis will have bladder pain.

    4. Examination of the ureters must precede the bimanual examination of the adnexa and ovaries because the vaginal fingers must push through the ureters lateral to the bladder and below the cardinal ligaments of the cervix in order to reach the ovaries. The ureter can be "plucked" by placing the vaginal fingers anterior and lateral to the cervix where the ureter passes laterally around the vagina and by compressing the ureter between the vaginal fingers and the abdominal fingers (lateral suprapubic). Pain stimulated by compression of the ureters should be referred to the groin on the same side in the area of where her abdominal wall triggerpoints are marked. 84% of women with interstitial cystitis and chronic pelvic pain will have ureteral pain.

    5. Examination of the uterus in the patient with a tender bladder can be accomplished by pressing the anterior uterus against the sacrum with the finger on the lower ureteral segment and cervix. The retroverted uterus can be palpated through the cul de sac and rectum. Cervical motion tenderness is of value in diagnosing peritonitis, but of little help in chronic pelvic pain. Only 15% of women will have a tender uterus with chronic pelvic pain.

    6. Palpation of the ovaries in the patient with bladder and ureter pain is difficult. In some patients it can be done with the rectal finger pressing laterally behind the uterus. The most accurate technique, however, is by pain mapping with diagnostic laparoscopy under conscious sedation where individual organs, adhesions, and endometriosis can be neurologically examined in the awake patient.

Symptoms of Pelvic Discomfort

Symptom	Cause	
1 Dysmenorrhea	a. Prostaglandin release during menses, resulting in uterine ischemia b. Retrograde bleeding c. Effect of prostaglandins on augmentation of pelvic neuropathic pain, i.e., interstitial cystitis d. Polyp or endometrial fibroid causing uterine contractions	40-60% women have PG dysmenorrhea which should respond to non-steroidals 15% do not respond to NSAIDs.
2 Predysmenorrhea	Cyclic pain that starts days to weeks before onset of bleeding due to changes in pain threshold affected by changes in estrogen and progesterone levels in patients with neuropathic pelvic visceral pain.	60% women with IC have predysmenorrhea
3 Dyspareunia	a. Neuroinflammatory pain of Bartholin's and Skene's glands with hypersensitivity of surrounding vestibular skin. Thinning makes vestibular skin more susceptible to tearing with coitus and irritation from yeast byproducts. b. Interstitial cystitis with both levator muscle and bladder tenderness c. Vaginal cuff neuromas	Most common cause of dyspareunia and most often misdiagnosed. 40-60% women unable to have intercourse

Causes of Acute and Chronic Pain

Diagnosis	% with diagnosis without pain	Mechanism of Pain	Induced referred somatic pain triggerpoints
1. Ruptured ovarian cyst	80-90%	Cyst fluid and blood potentiate sensitized peritoneum of peritoneal hyperalgesia	>90% of those with severe mittelschmerz will have triggerpoints
2. Adhesions	98%	Adhesions onto sensitized peritoneum may potentiate pain	Rare for cutting adhesions to alone cure pain
3. Ruptured ectopic pregnancy	<5%	Leukokinins from white cells in peritoneal blood sensitize peritoneum	Rare
4. Acute salpingitis	<1%	Endotoxins released by GC sensitize peritoneum tissue reaction to inflammation with bradykinin histamines, etc.	Rare
5. Endometriosis	60-80%	Infiltration of nerves by deep endometriosis sets up neuropathic pain reflex. Prostaglandins and infiltration of endometriosis in sensitized peritoneum potentiate pelvic pain.	70-90%
6. Adenomyosis	80-90%	Bleeding into myometrial muscle, release of prostaglandins increase menstrual cramps	<20%

7. Chronic pelvic pain	<5%	Dorsal horn sensitization of visceral, pelvic fascia, muscle, and somatic referral area by retrograde release of pain substances	95%
8. Interstitial cystitis	<10%	Dorsal horn sensitizes bladder pelvic muscles and somatic referral area by retrograde release of pain substances. Bladder changes secondary to pain substances include irritable muscles, thinning of transitional epithelium and loss of GAG layer, all resulting in increased pain.	60-80% Pain precedes bladder dysfunction

THERAPY

Treatment for neuropathic pelvic pain centers around two principles. Stop the things that hurt and alter the perception of pain.

- **Stopping the pain**

  a. Dysmenorrhea
     1) Birth control pills continuous
     2) Depo-Provera
     3) GnRH agonists
     4) Hysterectomy with or without oophorectomy

  b. Endometriosis
     1) Ablation or excision of lesions
     2) GnRH agonists
     3) Depo-Provera or continuous BCP
     4) Oophorectomy with or without hysterectomy

  c. Dyspareunia
     1) Outercourse
     2) Couples counseling
     3) Paracervical or vaginal cuff blocks
     4) Vestibulectomy
     5) Excision of vaginal cuff neuromas

  d. Painful bladder, interstitial cystitis
     1) Bladder diet
     2) Elmiron
     3) Hydrodilation
     4) Bladder cocktail

- **Altering the perception of pain**

  a. Tricyclic antidepressants

  b. Narcotics (short and/or long term)

  c. Anticonvulsants

d. Biofeedback

e. Physical therapy

f. Psychologic therapy

g. Nerve stimulus therapy
   1) Dorsal column stimulation
   2) Sacral nerve stimulation

h. Intrathecal pumps

i. Other nerve blocks
   1) Pudendal
   2) Epidural
   3) Differential spinal
   4) Hypogastric

j. Nerve interruption
   1) Uterosacral nerve ablation
   2) Presacral neurectomy

- **Other therapy protocols**

a. Interstitial cystitis

b. Irritable bowel syndrome

c. Chronic fatigue syndrome

d. Fibromyalgia syndrome

REFERENCES

Slocumb JC: Chronic somatic, myofascial and neurogenic abdominal pelvic pain. Clin Obstet Gynecol 1990;33:145-53. (Level III)

American College of Obstetricians and Gynecologists. Guidelines for women's health care. 2nd ed. Washington, DC: ACOG; 2002. (Level III)